AN ANALYSIS OF CLINICAL AND HISTOPATHOLOGIC CORRELATION OF SKIN TUMOURS

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CERTIFICATE

This is to certify that this dissertation on "**AN ANALYSIS OF CLINICAL AND HISTOPATHOLOGICAL CORRELATION OF SKIN TUMOURS"** is a work done by **S. SRI GAYATHRI**, under my guidance during the period 2003-2006. This has been submitted in partial fulfillment of the award of M.D.Degree in Pathology (Branch-III) by the Tamil Nadu Dr.M.G.R.Medical University, Chennai.

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INTRODUCTION

Skin is a heterogenous organ in which precisely regulated, cellular and molecular interaction govern many crucial responses to our environment(44). It is composed of varied elements having ectodermal and mesodermal origin. Most of these individual elements are capable of producing tumours and thus the number of different skin tumours exceeds that of any other organ(9).

Cutaneous neoplasm comprises an extremely diverse and sizable collection of pathologic entities. They may be divided into number of categories, reflecting their different biologic behaviors. These include hamartoma, reactive hyperplasia and benign tumours.

Virtually every individual will serve to a number of tumours in lifetime but majority of these are of little consequence and may never attract attention of the patient or his physician. In contrast to vast majority of inconsequential skin tumours, which may be passed off as birthmarks or blemishes, one may also encounter small group of serious malignant tumor originating in skin and capable of causing death(9).

Common experience suggests that most people suffer from one or more benign tumours such as melanocytic nevi, skin tags and seborrhoeic keratosis. The exact prevalence of benign tumours is unknown. But their frequency is much greater than hospital or private statistics would suggest. Most of the sources of information on tumours are based on selected series of cases seen in the clinic or by pathologists and cannot be related to the population from which they were drawn. The best epidemiological approach should be to examine the medical histories of all patients treated in geographically and demographically defined areas during a known period. Because skin lesions are visible and easily accessible, skin cancers provide us

with an excellent in vivo model to study the development of cancers.

Malignant tumours of skin constitute an important public health problem despite their low mortality rate. In most instances the histopathologic diagnosis is straightforward, very occasionally, tumours encountered are difficult to classify because of apparent morphological overlap with various appendageal tumours or the tumor exhibits both basaloid and squamous differentiation.

In addition to malignant tumours, one finds in the surface epidermis, so called **premalignant conditions** better regarded as tumours largely in situ. Although cytologically malignant, they are biologically still benign.

Multiple factors are involved in etiology of skin tumours. Genetic, environmental, racial, enzyme defects, radiations, geographic factors, exposure to sun rays are some of the factors implicated as etiology (47,24).

Genetic factors

Some of the skin tumours are dominantly inherited. These may be symmetrically distributed and most profuse on head and trunk. They may not manifest until puberty. Examples are Trichoepithelioma, Cylindroma, Steatocystoma multiplex, Basal cell nevus and Neurofibroma.(9,16)

Environmental factors

Sir Percivial Pott described the classic example of environmental causes in his work on cancer of scrotum in chimneysweepers. A number of other environmental causes have been attributed subsequently like coal tar, cresolite oil, mineral oil, crude paraffin, arsenic, and sunlight, X rays. UVB rays (290 – 320 nm) are the most damaging part of sunrays (5). The effect of sunlight is cumulative and produces other changes before malignancy occurs.

Exposure to ionizing radiations may occur as an accidental occupational hazard. Longer wavelength radiant energy and grey rays may be carcinogenic. Basal cell carcinoma is the most common cancer by ionizing radiation(17).

Xeroderma Pigmentosum is a genetically determined defect where the ability to repair the DNA after UV exposure is compromised. In these patients there is a high risk of light induced malignant skin tumours developing at an early age.

Cutaneous pigmentation confers a high degree of protection against light induced degenerative changes like solar keratosis, Squamous cell carcinoma and Basal cell carcinoma.

The malignant transformation may be attributed to the acquisition of additional genetic events or to immunosuppression due to an underlying neoplastic disease. Therefore patients with systemic diseases or malignancy should be carefully examined and followed for sudden changes in preexisting benign cutaneous tumours.

Neoplasms of skin comprise a wide spectrum of benign and malignant tumours that exhibit morphological differentiation towards one or more of structures found in normal skin.

Cutaneous adnexal tumours are a large and diverse group of tumours that are commonly classified according to their state of appendageal differentiation: eccrine, apocrine, follicular and sebaceous . These tumours generally behave in a benign manner, but malignant types exist.

Most tumours are relatively uncommonly encountered in routine practice, and pathologists can recognize a limited number of frequently encountered tumours. **In this study the histological features of selected but important benign and malignant tumours and tumor like lesions are discussed with emphasis on diagnostic approach and pitfalls in histological diagnosis.**

AIMS AND OBJECTIVES

The aims and objectives of the present study are,

1. To study the incidence of cutaneous tumours of clinical subjects reported to various out patients department in Kilpauk Medical College Hospital.

2. To determine the pattern of skin tumours in selected hospitals attached to Kilpauk Medical College in Chennai.

3. To study the relevance of Histopathological diagnosis with clinical correlation.

4. To study in detail the problems in diagnosis of cutaneous tumours.

REVIEW OF LITERATURE

Much of the literature on the subject of skin tumours, in relation to carcinoma is in cancer clinics or at population surveys (47,24). In this section, a comprehensive review of literature pertaining to various skin tumours encountered in the pathology Department of Kilpauk Medical College during the period of study is presented.

Skin tumours occur in a major group of dermatology patients. In a study at USA during the period of 1971-1974, 5.6 % of patients presented with skin tumours in various centers (47).

Classification of skin tumours

More than 150 types of skin tumours have been recognized. There is no uniformity in classification. WHO study group proposed a classification system in 1974. Elson B Helwig modified this classification in 1976.

The fundamental divisions are keratinocytic, melanocytic, appendageal, hematolymphoid and soft tissue tumours. WHO study group proposed a classification system in 1974. Elson B Helwig modified this classification in 1976.

Keratinocytic tumours

Among the carcinomas of skin, Basal cell epithelioma, Squamous cell carcinomas and Malignant Melanoma are common tumours. Carcinomas of skin occur predominantly on exposed areas. **In general, it is of low-grade malignancy and grows slowly with late metastasis to regional lymph node. Therefore the rate of curability is high in comparison with that of internal malignancy.**

Percival Pott was likely the first person to describe the malignant nature of squamous cell carcinomas in 1775. His description of “soot warts” in adolescent chimney sweepers let to the early appreciation of occupational carcinogen and promulgation of original occupational safety laws.

- Incidence of malignant skin tumours has been steadily increasing. In USA from 1932 – 1952, Squamous cell carcinoma and Basal cell carcinoma account for 0.06 and 0.18 % of hospital admission. (15)

- Multiple carcinomas of skin are reported to occur in 20 % of cases by Watson (43).

- In a largest study of its kind conducted from ten metropolitan areas in USA, Cutaneous carcinomas accounted 13.3 % of newly diagnosed cancers. (75).

- Cutaneous carcinomas, which develop in response to chemical irritation, are Squamous cell carcinoma (71).

- Incidents of malignancy that follow burns is one in five hundred, as observed by Pack and Trevas(72)

- Ward and Hendrick found that 60 % of skin carcinoma occurs in people over 60 years old. Both basal cell and Squamous cell carcinoma occur more frequently in men than in women.(74)

- The statistics from NewYork hospital medical centre over a period of 16 years 23120 were diagnosed as neoplasm. Among these 1.12 % was nevi, 2.36 % hemangioma and

lymphangioma, 3.76 % malignant tumours of skin(13).

- Pack and L Lever found that the average age of Basal cell carcinoma was 63 years (52).

- In indian literature, one of the early studies analysed the malignant tumours over a seven year period and found that out of 3083 malignant tumours, 132(4.28%) were skin tumours in which 81 (55.3%) were Squamous cell carcinoma, 23 (17.4 %) were Basal cell carcinoma, 20 (15.1%) were malignant melanoma, 3(2.2%) were Trichoepithelioma (59).

- During a three year study period, cancer of skin accounted for 1.87 % of all malignancies studied by Chakravathy(10).

- The ratio between Squamous cell carcinoma and Basal cell carcinoma was 3.9: 1(73,43).

- A large scale study by Paymaster et al concluded that during a period of 25 years the malignant neoplasm of skin constituted 1.5 % of all malignancies (54)

- In a study at CMC Vellore, there were 479 cases of primary tumours if skin, which comprised of Squamous cell carcinoma 39%, Basal cell carcinoma 16%, Nevi 11%, Melanoma 10% Benign adnexal tumours 10%, Benign epidermal tumours 8% and Malignant adnexal tumours 6%.(1).

Appendageal tumours

The numbers of current classification of appendageal tumours of skin indicate how little consensus exists about their grouping and their etiology and pathogenesis.

- Vaishnav et al in 1974 studied adnexal tumours of skin 48 (6.1%) adnexal tumours were seen in 783 tumours and tumor like lesion of skin over 12 years period (73).

- Venkateshvara Rao reported three cases (2.2%) trichoepithelioma in their study of neoplasm of skin.(69)

- Solanki et al during a period of 28 years observed 94 cases of skin appendage tumours. There were 50 (53.2%) cases of sweat gland tumours and 22 (23.4%) sebaceous gland tumours and 22 (23.4%) hair follicle tumours. (63).

  Review of literature pertaining to adnexal tumours seen in the present study group is given. (24,71,52,54,1,34,48)

- Ancel first reported cylindroma and described it as as turban tumor in1842. It was later reported and termed as endothelioma captis, neoepithelioma adenoids, tomato tumor, etc.

- Werth described Hidradenoma papilliferum in1872

  Mcdonald later reported apocrine origin (1941)

  Copper and Mcdonald described perianal localization (1944)

- Malharbe and Chemantais first described in1880 – Pilomatrixoma

  King described it as mummified epidermal cyst

  Mullet finally coined the term Pilomatricoma

Helwig and associates reported series of 500 cases with mean age of 29 years.

- Brooke and Fordyce reported the first case of Epithelioma adenoids cysticum in 1892

  Grey and Helwig later reported a study of 109 lesions in 50 patients

- R. Robinson described Hidrocystoma in 1893

  Baselline described Steatocystoma multiplex

  Pringle later coined the term Steatocystoma multiplex in 1899.

- Jarish coined the term Trichoepithelioma in 1894.

- Hirschfield first described Senile Sebaceous adenoma in 1904

  –Detailed review of it by Ranos, Silver and Portugal in 1953.

- Werther described Syringo cystadenoma papilliferum in 1913

  Helwig and Hackney later reported and reviewed 100 cases from 3 to 61 years.

- Meischer coined the term Trichofolliculoma in 1944

  Hymon and Clayman depicted histological aspects

  Gray and Helwig reported Trichofolliculoma in 32 cases

- Winer coined the term Dilated Pore and described the first case in 1954

- Hazel j, Vermon *et al*., reported an association of lung carcinoma with multiple cutaneous cylindromas.in 1988

- Tkahiro Sato and Mochio Katsumata in 1989 Ackermann studied formation of pearls and horn pearls of squamoid cells in detail and Reddy V B in 2000 regarded trichoepitheliomas as poorly differentiated hamartomas of hair germ.

Melanocytic tumours

- The original descriptions of melanoma of the hand, foot, vulva, and metastatic melanoma to the heart and bowel were by Jean Cruveilhier in his Anatomie Pathologique du Corps Humans published between 1829 and 1842

- Rippey JJ, studied relationship of moles and melanoma in 1977(63).

- Maize JC in 1984 studied the histologic association of Dysplastic melanocytic nevi with primary cutaneous melanomas (95).

- Clark J H 1984 studied Six evident lesional steps of tumor progression form the neoplastic system that affects the human epidermal melanocyte: 1) the common acquired melanocytic nevus; 2) a melanocytic nevus with lentiginous melanocytic hyperplasia, i.e., aberrant differentiation; 3) a melanocytic nevus with aberrant differentiation and melanocytic nuclear atypia, i.e., melanocytic dysplasia; 4) the radial growth phase of primary melanoma; 5) the vertical growth phase of primary melanoma; and 6) metastatic melanoma.(11)

- Rhodes AR. Studied in 1986 about the precursors include lentigo maligna, dysplastic melanocytic nevi, congenital nevi (of any size), and darkly pigmented lesions of acral surfaces and mucous membranes (61)

- Jimbow K, in 1991 discussed the role of The epidermal melanin unit in the pathophysiology of malignant melanoma(33)

- Byers HR in 1998 Histologic criteria may never be completely sufficient to predict behavior accurately, because the fundamental change that renders a cell aggressive may

not be morphologically reflected and may require immunohistochemical or other molecular markers to establish behavior (7)

- Scolyer RA in 2004 established that the dermal mitotic rate of a primary cutaneous melanoma is a major prognostic determinant, and have shown that its assessment and that of other important histopathologic prognostic variables are reproducible between pathologists. Sentinel node (SN) biopsy has provided a minimally invasive procedure that can accurately predict the regional node status of melanoma patients(65)

- Hussein MR. In 2005 studied that melanocytic dysplastic naevi occupy the middle ground between benign melanocytic naevi and cutaneous malignant melanomas (30).

- Rager EL in 2005 reported that Melanoma is an increasingly common malignancy, and it affects a younger population than most cancers. Risk factors for melanoma include white race, sun sensitivity, family history of melanoma, and melanocytic nevi. Sunburn and intermittent sun exposure appear to increase the risk of developing melanoma (57).

Hematolymphoid tumours

- Williamze R, kerl H et al proposed EORTC classification of primary Cutaneous lymphomas(78)

- Burg G et al proposed revised WHO/EORTC classification of Cutaneous lymphomas 2005 (6).

Soft tissue – Vascular tumours

- Johnson et al in 1976 discussed the pathology of Cutaneous vascular tumours(34).

- Requena et al in 1997 classified cutaneous vascular anomalies into the following categories: hamartomas, malformations, dilatations of preexisting vessels, hyperplasias,

benign neoplasms, and malignant neoplasms(60)

- Hervella et al indicated that Haemangiomas could be indicators or clue signs for serious syndromes. Although less well known than those related to vascular malformations, there are some syndromes of important diagnostic value that are associated with haemangiomias(25)

Soft tissue – Fibrous tumours

- Manchini et al in 1962 described the histogenesis of experimentally induced keloids(46)
- Neisser.F.B. in 1999 described the nature of keloid and hypertrophic scar (49)
- Shaff, Taylor described and analysed Histologic features of keloidal scars in 2002. (66)

Soft tissue – Smooth muscle tumours

- Calonje, Raj.S. Did a clinicopathologic analysis of 53 lesions of Cutaneous pilar tumours in 43 patients (55)
- Mathews in 2004 reported cytologic atypia in cutaneous leiomyoma similar to symplastic leiomyoma(48)
- Meitterer in 2006 evaluated the biologic potential of Cutaneous leiomyomas.(49)

MATERIALS AND METHODS

We have reviewed the clinical records of patients from the Department of pathology, Govt Kilpauk medical college who were diagnosed with skin tumours by histopathology between 2004 and 2006. Recurrences and diagnostic duplicities (tumours first biopsied and then

excised) were excluded.

Specimens were received from Refferal centres like Govt. Medical college the hospital in department of Dermatology, Surgery, Plastic surgery, which formed the major source of specimens in this study period. Specimen from government Royapettah hospital, Government hospital for thoracic medicine, Chetpet, Thiruvallur and private hospitals were also included in the study.

All biopsies were taken from grossly characteristic areas. Multiple biopsies were advised when lesions present in differing forms and stages. Technique used in skin biopsy is of prime importance in the diagnosis of any skin disease.

FOUR TECHNIQUES ARE EMPLOYED

1. Scalpel biopsy

This method is used in removal of a typical pigmented lesion and subcutaneous nodules. Incisional scalpel biopsies are used when adjacent normal areas are to be studied and in cases of panniculitis.

2. Punch biopsy

This is used to obtain samples from inflammatory dermatoses, scalp lesions. Punch biopsies should be ideally 3 mm to 4 mm.

3. Shave biopsy

This method is used in lesions in which histological changes are to be present in epidermis or superficial dermis. It cannot differentiate between squamous cell carcinoma and keratoacanthoma. It cannot assess the depth of invasion in melanoma.

4. Curettage biopsy

It is the least satisfactory method for obtaining material for Histologic examination because the submitted material is usually scanty and superficial. It has lost its architecture and may show crush artifact.

Each specimen was given a number, detailed clinical history like age, sex, occupation, family history, presentation and history of sun exposure, history of previous therapy, and other investigation details were noted for analysis (Appendix 1).

Macroscopic gross details of the specimen were noted down and bits were taken respectively. **Inking of margins was done whenever necessary to know the extent of infiltration of the lesion.** Sections were made from paraffin embedded specimen and stained with Hematoxylin and Eosin routinely. Special stains were done as and when necessary. Fine Needle Aspiration Cytology was done on palpable lesions biopsy correlation was obtained.

These tumours were classified according to world Health Organization's International Histological Classification for Skin Tumours and were tabulated.

Different types of tumours were observed, which are arranged in five groups according to WHO classification. (55) This is shown in table 3.

I a Keratinocytic tumours

Benign lesions

Seborrhoeic keratosis

Keratoacanthoma

Premalignant lesions

Bowen's disease

Actinic Keratosis

Malignant lesions

Basal cell carcinoma

Basal cell carcinoma with adnexal differentiation

Basosquamous carcinoma

Squamous cell carcinoma

Verrucous squamous cell carcinoma

I b Tumor like lesions

Dermoid cyst

Epidermal cyst

Pilar cyst

Sebaceous cyst

Trichilemmal cyst

Epidermal nevus

II Melanocytic tumours

Malignant melanoma

Melanocytic nevi

Common blue nevi

III Appendageal tumours

a. Benign tumours with apocrine and eccrine differentiation

Hidrocystoma

Syringoma

Hidradenoma

Spiradenoma

Syringocystadenoma papilliferum

Mixed tumour (chondroid syringoma)

b. Benign tumours with follicular differentiation

Pilomatricoma

Tricholepithelioma

c. Malignant tumours with follicular differentiation

Proliferating trichilemmal tumour

d. Tumours with sebaceous differentiation

Nevus sebaceous

IV Haematolymphoid tumours

Cutaneous lymphoma

V Soft tissue tumours

a. Vascular tumours

Pyogenic granuloma

Cavernous haemangioma

Angiokeratomas

b. Smooth muscle tumours

Cutaneous leiomyoma

c. <u>Neural tumours</u>

Neurofibroma

Miscellaneous

Cutaneous metastasis

Calcinosis cutis

OBSERVATION AND RESULTS

INCIDENCE

During the study period of three years, (June 2004 – June 2006) 14,259 specimens were received in the department of pathology, Kilpauk medical college. Of these, **3251** (22.8%) specimens were neoplastic lesions and of these 200 (6.13 %) were skin tumours.

The Age distribution is given in the table 1.

TABLE – 1

AGE DISTRIBUTION

Age Group	Number	Percentage
0-20	34	17
21-40	75	37.5
40-61	69	34.5
>61	22	11
Total	**200**	**100**

It could be inferred from the table 1 that 17 % of patients belong to age category of below 20 years, 37 % between 21 and 40 years, 34.5 % between 41 and 60 years and the remaining 11 % above 61 years.

TABLE – 2

SEX DISTRIBUTION

Sex	Number	Percentage
Male	105	52.5
Female	95	47.5

There were 52.5 % (105) male patients, 47.5 % (95) were female patients.

TABLE – 3

NATURE OF TUMOR

Nature	**Number**	**Percentage**
Benign	162	81
Malignant	38	19
Total	**200**	**100**

The tumours were grouped into Benign and Malignant based on Histopathlogical Diagnosis. Benign tumours accounted for 81% of tumours and Malignant tumours accounted for 19%.

TABLE – 4

AGE Vs NATURE OF TUMOR

	Number	**Mean age**	**95% CI**	**Ucl of Mean**	**P value**
			LcL of Mean		
Benign	162	35.00823	32.579	37.4367	p= 0.000
Malignant	38	56.763116	52.34705	61.17926	Significant

The average age in which Benign and Malignant tumours were grouped was studied. Benign tumours were mostly seen in the age 33-38 years and malignant tumours in the age 52-61 years and statistically significant.

TABLE - 5

SITE OF OCCURRENCE AND MORPHOLOGY OF LESIONS

Site	Nodule	Papule	Plaque	Wart	Macule	Ulcer	Total
Scalp	20 (16.39%)	5 (35.71%)	3 (25.00%)	2 (7.41%)	1 (6.25%)	2 (22.22%)	33 (16.50%)
Face	53 (43.44%)	8 (57.14%)	5 (41.67%)	8 (29.63%)	9 (56.25%)	1 (11.11%)	84 (42.00%)
Trunk	16 (13.11%)	1 (7.15%)	-	7 (25.93%)	1 (6.25%)	4 (44.45%)	29 (14.505)
Extremities	33 (27.06%)	-	4 (33.33%)	10 (37.03%)	5 (31.25%)	2 (22.22%)	54 (27.00%)
Total	122	14	12	27	16	9	200
% to Total	61	7	6	13.5	8	4.5	100

The site of occurrence of the tumours and their morphology were analysed It could be observed from the table 5, that 16.50 % of lesions presented on scalp, 42 % on face, 14 % on trunk, 27 % on extremities. Nodular lesions and ulcerative lesions were usually single lesions and macular lesions and papular lesions were usually multiple. Benign lesions occur in the form of nodules, papules or macules. Ulcerative lesions were almost malignant. Of the skin tumours, 61 % presented as nodules, 7 % as papules, 6 % as plaque, 13.5 % as wart, 8 % as macules and 4.5 % as ulcer. The lesions present as nodules were mostly benign and lesions present, as ulcers were mostly malignant.

TABLE - 6

MORPHOLOGY OF BENIGN AND MALIGNANT LESIONS

	Nodule	**Papule**	**Plaque**	**wart**	**Macule**	**Ulcer**	**Total**
Benign	107 (66.05%)	12 (7.42%)	9 (5.55%)	18 (11.11%)	14 (8.64%)	2 (1.23%)	162
Malignant	15 (39.47%)	2 (5.26%)	3 (7.89%)	8 (21.05%)	2 (5.26%)	8 (21.05%)	38

It could be inferred from the table 6 that Nodular lesions were seen in both benign and malignant lesions. Ulcerative lesions were rare in Benign tumours and were more commonly seen in malignant tumours. Macules, papules, warts and plaques were found in both Benign and Malignant tumours.

Number of Patients with excess sunlight exposure was studied based on their occupation, and was correlated with various types of tumours. Patients with excess sunlight exposure accounted for 27.5%.

TABLE – 7

SUNLIGHT EXPOSURE

	Number	**Percentage**
Yes	55	27.5
No	145	72.5

TABLE – 8

NATURE OF TUMOUR AND SUNLIGHT

	Yes	No	Total
Benign	32	130	162
Malignant	23	15	38
	55	145	200
	p value= 0.0000		

Out of 162 patients with benign tumours 32 patients (19.75%) had excess sunlight exposure and out of 38 patients with malignant tumours 23 (60.52%) of them had excess sunlight exposure.

Different types of tumours were observed, we follow WHO classification to arrange them in groups. **TABLE - 9**

SAMPLE ANALYSIS AMONG VARIOUS GROUPS

Type of Neoplasm	Total Number of cases out of 200	Percentage
Keratinocytic	106	53%
Appendageal	29	14.5%
Melanocytic	27	13.5%
Hematolymphoid	02	1%
Soft tissue		
i) Fibrous	08	4%
ii) Smooth muscle	02	1%
iii) Vascular	18	9%
Neural	07	3.5%
Miscellaneous	01	0.5%
Total	200	100

Based on the WHO classification, the sample was categorised into groups as given in table 9. It could be inferred that 53 % of the total sample were keratinocytic, 14.5 % were

appendageal, 13.5 % were melanocytic, 1 % Hematolymphoid tissue, 9 % were vascular soft tissue, 3.5 % were neural and 0.5 % were miscellaneous lesions including metastasis and calcinosis cutis.

It could be inferred from the table 4 that keratinocytic tumours show a male preponderance, appendageal, melanocytic and neural tumours show a female preponderance. **Keratinocytic tumours were seen more commonly in the age group of 41 to 60 years, appendageal tumours in the age group of 21 to 40 years and melanocytic tumours in the age group of 41 to 60 years.**

Benign tumours were

Appendageal tumours 93.10%

Melanocytic tumours 77.18%

Keratinocytic tumours 73.58%

Hematolymphoid tumours 50%. Almost all of soft tissue tumours were benign.

Malignant tumours were seen in

26.42% of keratinocytic tumours

22.2% of melanocytic tumours

50% of hematolymphoid tumours.

6.90% of Appendageal tumours

One case of cutaneous metastasis was also observed in the study in which primary was

found in Colon. Malignant tumours included in the present study are Squamous cell carcinoma (21), basal cell carcinoma (12), Malignant melanoma (6), Trichoblastic carcinoma(1) and these account for 39 (19.5%) of all Cutaneous tumours.

TABLE – 10

SITE Vs TYPE OF TUMOR

Site	Keratinocytic	Adnexal	Melanocytic	soft tissue	Hematoly-mphoid	Total
scalp	14	6	1	11	1	33
Face	45	15	16	7	0	83
Trunk	8	0	1	3	0	12
Extermities	39	8	9	16	0	72
Total	106	29	27	36	1	200

The site of occurrence of lesions were also studied and tabulated in the table. 10. The results would show that Head and neck region involved in

51.74% of Appendageal tumours

43.4% of keratinocytic tumours, and

16.12% of melanocytic tumours

Scalp is involved in

50% of Hematolymphhoid tumours, and soft tissue vascular tumours

20.69% of Appendageal tumours, and

13.2% of keratinocytic tumours .

Upper and lower extremities were involved by

33.3% of melanocytic tumours

27.59% of Appendageal tumours

22.6% of Keratinocytic tumours,

7.14% of soft tissue fibrous and neural tumours.

Keratinocytic tumours

Keratinocytic tumours constitute 53 % of total neoplastic lesions of which 11 were benign, 33 were malignant, 2 were pre-malignant and 60 were tumor like lesions. Among the tumor like lesions commonest is Epidermal cyst which occur more often in face, next common is Trichilemmal cyst, which is commonly seen in scalp.

It could be evident from the tables 9 and 10 that these tumours showed male predominance and increased frequency among the age group 41 to 60 years. These are mostly benign tumours that present study, 43.4 % of keratinocytic tumours occur in face, 20.75 % in trunk, 22.64 % in extremities and 13.2 % in scalp. 64.15 % of them are nodular lesions, 5.6 % papules 6.6 % are plaques, 16.9 % warty lesions and 5.67 % ulcerative lesions. Ulcerative lesions are usually malignant and benign lesions present as nodules.

TABLE - 11

KERATINOCYTIC TUMOURS

Malignant tumours	Number of cases	% to total
Basal cell carcinoma	12	11.3%
Squamous cell carcinoma	21	19.8%
Pre malignant lesions	2	1.88%
Actinic keratosis	1	
Bowen disease	1	
Benign tumours	11	10.37%
Seborrheic keratosis	3	
Keratoacanthoma	1	
Squamous papilloma	7	
Tumor like lesions	60	56.60%
Dermoid cyst	7	
Epidermal cyst	40	
Pilar cyst	2	
Steatocystoma	1	
Trichilemmal cyst	9	
Epidermal nevus	1	
Total	**106**	**100.00**

Appendageal tumours

Appendageal tumours make up 14.5% of the study group, 27 of them are benign and 2 are malignant. 15 of these tumours were derived from eccrine and apocrine glands, 11 of them

showed follicular differentiation, and 3 of them showed sebaceous differentiation. As seen in the tables 4-7, appendageal tumours are mostly single lesions. They show a maximum occurrence in face as nodular and popular lesions. Tumours of follicular differentiation are multiple papular lesions more often seen in face. The malignant. Appendageal tumours are two in number with follicular differentiation.

TABLE - 12

APPENDAGEAL TUMOURS

Appendageal tumours	Number of cases	% to total
Benign tumours with apocrine and eccrine differentiation		
i) Hidrocystoma	1	3.45
ii) Syringoma	1	3.45
iii) Hidradenoma	5	17.24
iv) Eccrine poroma	1	3.45
v) Spiradenoma	1	3.45
vii) Mixed tumour (chondroid syringoma)	2	6.90
Benign tumours with follicular differentiation		
i) Pilomatricoma	3	10.34
ii) Trichoepithelioma	6	20.69
Malignant tumours with follicular differentiation		
a) Proliferating tricholemmal tumour	1	3.45
b) Trichoblastic carcinoma	1	3.45
Tumours with sebaceous differentiation		
Nevus sebaceous	3	10.34
Total	**29**	**100.00**

Melanocytic tumours

There were 27 Melanocytic tumours, which constitute 13.5 percent of total neoplasms. Common acquired melanocytic nevus comprises 74 % of the melanocytic lesions. These tumours

commonly occur in face as asymptomatic macules or papules. 22.2 percent lesions were malignant. Malignant melanoma was observed in extremities as nodular or warty symptomatic lesions. Two of six cases had metastases in inguinal node.

Of all the melanocytic tumours, 74.07 % were common melanocytic nevus, 22.22 % were malignant melanoma and 3.70 % were blue nevus.

TABLE - 13

MELANOCYTIC LESIONS

Melanocytic lesions	Number	Percentage
Common melanocytic nevus	20	74.07
Blue nevus	1	3.70
Malignant melanoma	6	22.23
Total	27	100.00

Hematolymphoid tumours

Two hematolymphoid tumours were observed in the study, both were in male, one was found in scalp, other was in extremity. All the hematolymphoid tumours were cutaneous lymphoma.

Soft tissue tumours –Fibrous

Fibrous soft tissue tumours were eight in number, with a female predominance, these tumours were commonly found in extremities as nodules in the age group between 21 and 60.

TABLE - 14

SOFT TISSUE TUMOURS-FIBROUS

Soft Tissue Tumours-Fibrous	Number	*Percentage*
Cutaneous fibrous histiocytoma	1	12.50
Fibroma	5	62.50
Keloid	1	12.50
DFSP	1	12.50
Total	8	100.00

Soft tissue – vascular

Vascular soft tissue tumours were eighteen in number that make up 9 percent of the total sample. These tumours were single tumours with male predominance. More often found in scalp (50%). They commonly presented as nodule(77.55) and 50% are found in the age group 21- 40 years.

TABLE - 15

SOFT TISSUE VASCULAR

Soft Tissue Vascular	Number	%
Capillary angioma	11	61.10
Cavenous hemangioma	3	16.67
Glomus tumor	1	5.56
Granular pyrogenicum	3	16.67
Total	18	100.00

Soft tissue – smooth muscle

Two cases of cutaneous leiomyoma were observed, one was seen in the trunk and the other was found in leg. With equal male female distribution in the age group 21 to 60.

Neural tumours

Seven tumours (3.5%) with neural differentiation were observed in the study . They showed a male predominance. All were benign lesions mostly in extremities as nodular lesions. Four of them were single and three were multiple.

Miscellaneous

One case of metastatic deposit of adenocarcinoma colon was observed in a male patient of 69 years.

One case of anaplastic carcinoma in scalp was observed in 45 years male.

DISCUSSION

The true incidence of skin cancer is difficult to measure exactly as unknown number of patients may never seek medical advice, because the skin cancer is disease of elderly people, who may have important concerns other than a nodule in skin.

14,259 specimens were received in the pathology department of Kilpauk Medical College, Chennai city of these 3251(22.8%) were neoplastic lesions and of these 200 were skin tumours giving a percentage of 6.13%.

Of these benign tumours constituted 81% and malignant tumours accounted for 19% of total lesions.

TABLE - 16

COMPARISON WITH SIMILAR STUDY IN 1986(56)

	Zhonghua Zong et al-1986	**Present study 2004-2006**
Total number of cases	32425	200
Benign tumours	2137(62.4%)	162(81%)
Malignant tumours	1288(37.6%)	38(19%)
Benign Epidermal tumours	1742(81.5%)	78(39%)
Benign Appendageal tumours	395(18.5%)	27(13.5%)
SCC	958(74.4%)	21(10.5%)
BCC	296(23.0%)	12(6%)
SCC:BCC	3.2:1	1.75:1
malignant appendage	34(2.6%)	2(1.0%)
age difference	Benign tumours in adolescent and adults, Malignant tumours in elderly	Benign tumours in adolescent and adults, Malignant tumours in elderly
Site difference	Mostly in head and neck	Mostly in head and neck region
Sex ratio	1.5:1	1.1:1

Our study did not reveal any occupational predisposition to skin tumours. Most of the patients with malignant tumours had any environmental predisposition described others

(16,17), except one patient who developed Squamous cell carcinoma following Burns Scar giving a percentage of 8.3% against less than 1% in the study by Pack and Trevas.

Clinical diagnosis compared with histological diagnosis for benign and Malignant tumours. Sensitivity and Specificity obtained.

Of the various cutaneous tumours in the present study, 38 cases were malignant lesions.

Squamous cell carcinoma 51.21%

Basal Cell Carcinoma 29.26%

Malignant melanoma 14.6%

Malignant Adnexal tumor 4.8%

This when compared with a study in 1995 on Trends of skin cancer in Canton of vaud (41).

TABLE - 17

COMPARISON WITH A STUDY IN CARTON OF VAUD

	Skin Cancer in Vaud Levie. F. et al (1995)	**Present study (2007)**
Study period	1972-1992	2004-2006
Total no of cases	12473	200
Sqamous Cell carcinoma	25%	51.21%
Basal cell carcinomna	63%	29.26%
Malignant Melanoma	9%	14.6%

KERATINOCYTIC TUMOURS

The keratinocytic tumours are a clinically and histopathologically diverse group of lesions derived from the proliferation of epidermal and adnexal keratinocytes. At one end of the spectrum the proliferations are benign (acanthomas) and usually of cosmetic importance only, while at the other there are malignant tumours, which uncommonly may be aggressive with

metastatic potential, as seen with some squamous cell carcinomas. Included in the spectrum are the epidermal dysplasias (actinic keratosis, arsenical keratosis and PUVA keratosis) and intraepidermal carcinomas (Bowen's disease and bowenoid papulosis). Keratinocytic tumours are important public health problem despite their low mortality (46). Keratinocytic tumours account for approximately 90% or more, of all skin malignancies (77).

Seborrheic Keratosis

These lesions are often multiple, oval, raised and pigmented. They have classic stuck-on appearance. The sudden increase or appearance in number and size of Seborrheic Keratosis is associated with internal malignancy known as Leser Trelat sign (3). Three cases (2.83%of all keratinocytic tumours) of Seborrheic Keratosis were observed in the study, both in the head and neck region.

Histologically the lesions showed hyperkeratosis, acanthosis, papillomatosis, of epidermis. The lower border is even and and a straight line could be drawn from one end of the lesion to the other end. Squamous eddies found in these lesion was differentiated from horn pearls of the squamous cell carcinoma by their large number, small size, and circumscribed configuration.

Actinic Keratosis

One case of actinic Keratosis in face was observed in the present study. In this portion of epidermis exposed to sunlight, a sequence of atrophic, hyperplastic and eventually dysplastic changes known as actinic or solar Keratosis may develop. Histologically the lesion shows alternating columns **of orthokeratosis and parakeratosis** with atypia in basal epithelium and in keratinocytes. Because there is not a sharp differentiation between Actinic Keratosis and

Squamous Cell Carcinoma in situ is not always possible to differentiate the two. In SCC, irregular aggregate of atypical keratinocyte will be found in papillary dermis not in contiguity with overlying epidermis(39) serial sectioning is essential in differentiating the two.

Bowen's Disease

One case of Bowen's disease in external genitalia was observed in the study. Clinically the lesion showed scaly, erythematous plaques. Histologically epidermis showed acanthosis with elongation and thickening of rete ridges. Throughout the epidermis the keratinocytes lie in complete disorder resulting in "**wind blown appearance**" with atypical individual cell keratinisation. No Histologic difference exists between Bowen's Disease and Bowenoid Actinic Keratosis(50).

Squamous Cell Carcinoma

12 cases of Squamous cell carcinoma were observed which constituted 11.32 % of all keratinocytic tumours. They were present mostly as solitary slow growing nodules with central ulceration in extremities. One case was found in the scalp following chronic inflammation, one was found in extremities following burns scar. Histologically lesions showed epidermal proliferation with full thickness cellular atypia, hyperchromatic nuclei, and absence of intercellular bridges, keratin pearl formation and zonal necrosis. Invasion into dermis is considered as sinequanon feature for diagnosis.

Verrucous Carcinoma

Verrucous Carcinoma is a low-grade squamous carcinoma. 9 cases of Verrucous Carcinoma were observed in this study, which constitute 8.49 % of the total keratinocytic

tumours. All were slow growing exophytic, verrucous tumours in foot. They bear a superficial resemblance to intractable wart, which show Histologically nuclear vacuolation and eosinophilic cytoplasmic inclusions they penetrate deep into the tissue. Histologically, these tumours show hyperkeratotic, acanthotic epidermis and the tumor invades with **broad strands that bulldozes into the deeper tissue**. (4) Nuclear atypia, individual cell keratinisation and horn pearls seen in squamous cell carcinoma were absent. Large deep biopsy is essential for diagnosis.

Keratoacanthoma

Keratoacanthoma is a variant of squamous cell carcinoma with a potential for spontaneous regression. One case of keratoacanthoma was observed in the study. It was a dome shaped nodule with horn filled crater in its centre. Keratoacanthoma was differentiated from Squamous cell carcinoma, inverted follicular Keratosis, Pseudoepitheliomatous hyperplasia and Verrucous carcinoma mainly by its architectural features and by cytologic findings

Basal Cell Carcinoma

12 cases of basal cell carcinoma were observed in the study. Of which one showed follicular differentiation, one was pigmented and one showed basisquamous elements. Pigmented variant was clinically diagnosed as Melanoma. Basal Cell Carcinoma accounted for 9.43 percent of all keratinocytic tumours and 6% of all skin tumours. All these tumours were observed as single lesions in the head and neck regions. 47 % were found in females and 53 % in males. They all belong to the age group of 40 to 60 years. Histologically, the tumours showed nests and islands of basaloid cells with pallisading at the periphery. Characteristic retraction artifact was observed between the pallisading cells and the stroma due to increase in basal lamina material. In a patient with nodule in face, FNAC was performed and reported as Basal Cell carcinoma with follicular differentiation. On biopsy the lesion was found to be Basal cell hamartoma

In a study by Ackerman squamous cell carcinoma was the common cancer in humans (3). The ratio of BCC and SCC in the present study is 1.75:1. This is comparable to many other studies in Basal Cell Carcinoma (15,75,69).

Tumor like lesions

Tumours like lesions were often observed in the epidermis, which were often mistaken for malignancy.

Epidermal Cyst

40 cases of epidermal cysts were observed in the study, which accounted for 37.74% of all keratinocytic tumours. Most of them were male. They were predominantly nodular lesions in hair bearing regions. The wall epidermal cysts showed true epidermis with a layer of granular cell and many layers of squames. Foreign body granulation tissue was observed in five of the cases due to release of the contents of the cyst into dermis.

Dermoid cyst

7 cases of dermoid cysts were observed in this study. They belong to the age group of less than 40 years with equal male and female ratios. The wall of the dermoid cysts were lined by epidermis with various fully mature appendages like hair follicles and sebaceous glands. (19)

Trichilemmal cysts

9 cases of Trichilemmal cysts were observed in this study. This accounted for 8.49% of all keratinocyttic tumours. Most of them were single and occurred in scalp. The wall of Trichilemmal cyst was composed of epithelial cells with peripheral pallisading, abrupt keratinisation and frequent calcification.

Steatocystoma

One case of streatocystoma was observed in a 40 years old female as multiple lesions in the labia majora. Cyst wall was lined by intricately folded several layers of flat cells,

eosinophilic horny layer with decapitation secretion(53), with lobules of sebaceous glands in dermis.

Appendageal tumours

Cutaneous appendageal tumours are a large diverse group oftumours that are commonly classified according to their state of appendageal differentiation- eccrine, apocrine, follicular and sebaceous. These tumours generally behave in a benign manner, but malignant types exist.

29 appendageal tumours were observed in the study. Of which 15 were sweat gland tumours, 3 were sebaceous gland tumours and 11 showed follicular differentiations.

These observations as compared with a previous study in 2003 on appendageal tumours in Agha khan University hospital by Yaqoob et al in Pakistan is given below(79).

TABLE – 18

COMPARISON WITH A SIMILAR STUDY IN AGHA KHAN UNIVERSITY

	Yaqoob et al (2003)	**Present study (2004-2006)**
Benign	87.3%	73.58%
Malignant	12.7%	26.42%
Male : Female ratio	1:1	1:1.6
Mean age	41.72	35.2
Eccrine and Apocrine differentiation	51.79%	51.72%
Pilosebaeceous differentiation	41.56%	48.28%
Common Benign Tumor	Pilomatricoma	Trichoepithelioma

Sweat gland tumours

Hidrocystoma

One case of hidrocystoma in the study showed single cystic cavity with two layers of small cuboidal cells.It presented as solitary bluish cystic nodule in neck region. Bluish colour is due to presence of lipofuschin, melanin, hemosiderin and Tyndall effect. (55) Eccrine secretory tubules and ducts were located below the cyst. Papillary extensions into the cyst cavity, observed by Smith J.D.(67), Single layer of cells observed by Hason Khan(40) were not observed in the present study.

Chondroid syringoma

Mixed tumor of skin is the term introduced by Hirsch and Helwig in 1961(58) has been replaced by the term Chondroid Syringoma. 2 cases observed in the study were firm intradermal nodules seen in face. Hirsch.P. (58) observed the chondroid syringomas to occur more commonly in head and neck. Similar observations were observed in the study. Histologically, tubular lamina with two layers of epithelial cells occurred in an abundant mucinous stroma with basophilic cartilage like areas. Headington J.T. (23) studied apocrine type of decapitation secretion in some mixed tumours of skin. Such observation was seen in one of the two cases in the present study. Malignancy was not evident Histologically.

Nodular Hidradenoma

Although rare(62) it is common among the adnexal tumours comprising 17.24% of all adnexal tumours. It is manifested in the 3 rd to 5^{th} decade as mentioned by Feldman et al(18). 5 cases of nodular Hidradenoma observed in the present study were intradermal nodular lesions in head and neck region presenting in the age group of 30-40 years. This was similar to the observation made by Efskind J Eker (34).

Spiradenoma

One case of Spiradenoma in the present study was a solitary intradermal nodule over the face. The tumor was highly cellular with sharply demarcated lobules containing intertwining cords of epithelial cells, surrounded by capsule f compressed connective tissue. Two types of cells were noted small basophilic cells at the periphery and large pale cells in the center. **The much-appreciated histological clue the perivascular spaces also observed in the present study** was first described by Van Den oord and Chris De wolf Peters in 1998 (36). It consists of variably sized spaces around one or more central blood vessels bordered at the periphery by a palisade of tumor cells and lined at both sides by basement membrane. The spaces are empty or filled by pink proteinaceous material.

Syringocystadenoma papilliferum

4 cases of Syringocystadenoma papilliferum observed were commonly found in the scalp and face. Histologically, epidermis showed cystic invaginations lined by keratinizing squamous cells in the upper part and papillary projections lined by two rows of epithelium in the lower part. The stroma was densely cellular with plasma cells. **Linear or zosteriform configuration** or malignant changes described in the literature were not evident in this study. There was no association with nevus Sebaceous which usually harbor 80% of its origin (12).

Eccrine poroma

One case of Eccrine poroma observed in the study was seen as firm raised asymptomatic nodule in forearm. Histologically, the tumor grows downward into the dermis as broad anastomosing bands of epithelial cells that are smaller than keratinocytes.

Tumours of sebaceous glands (5,8)

4 cases of nevus sebaceous were seen in the scalp, which is the common site. Histologically tumor showed proliferations of sebaceous glands, papillomatous epidermis and ectopic apocrine glands deep in the dermis.

Tumours with follicular differentiation

Histopathologically 33% of tumours show follicular differentiation, compared to 63.4% by Jayalakshmi et al (32) and 25% by Weigh et al(76).

Trichoepithelioma

6 cases of Trichoepithelioma were observed in the study. All were multiple lesions in the form of skin colored papules mainly if the nose and nasolabial fold. Single lesions (20) was not observed. Histologically, all of them showed horncyst surrounded by basophilic cells showing Trichilemmal keratinisation., except for one case where there was no horn cyst formation. Islands of basophilic cells show peripheral pallisading. Important histopathological differential diagnosis is Basal Cell Carcinoma and was differentiated by the absence of retraction artifact of stroma. **Papillary mesenchymal bodies, amyloid deposits, mitosis and inflammatory granuloma are other features that distinguish trichoepithelioma from Basal cell carcinoma.**

Pilomatricoma (Synonym: Calcifying Epithelioma of Malherbe)

Pilomatricoma are benign tumours with differentiation towards hair matrix account for 20% follicular tumours(38). In a review of 209 patients by Julien et al (37), the youngest patient was 18 months, oldest 86 years with bimodal age distribution.

3 case of Pilomatricoma observed in the study were found in face and upper extremities occurring more commonly in the age group of less than 20 years. This observation was similar to the observation by Moehlenbeck in his study on Pilomatricoma(72). Histologically, the tumor is composed of irregular shaped islands of two types of epithelial cells (basophilic cells and shadow cells) with calcification and foreign body reaction. Malignant transformation in pilomatricoma was not observed in the present study.

Proliferating trichilemmal tumour

One case of Proliferating trichilemmal tumour observed in the study was seen in scalp as an elevated lobular mass with ulcerations. Histologically, the tumor was composed of multiple lobules of squamous epithelium with peripheral pallisading. The tumor showed extensive areas of trichilemmal keratinisation, tissue invasion and presence of severe atypia and giant nuclei.

Trichoblastic carcinoma

One case of Trichoblastic carcinoma was observed in the present study. Histologically, it was made of proliferation of hair germ cells in the form of islands of basaloid cells with peripheral pallisading, occupying the dermis and infiltration into subcutaneous fat. They showed moderate degree of cytological atypia. It was differentiated from basal cell carcinoma by lack of clefting artifact, stromal edema and ulceration.

Melanocytic lesions

Melanocytic proliferations are composed of one or more of three types of cells; Melanocytes, Nevus cells and Melanoma cells. Each of which may be located in the epidermis

or dermis.

Benign melanocytic tumours include various types of nevi Most of the benign pigmented lesions are asymptomatic and are not excised. They were usually biopsied for cosmetic reasons and fear of melanoma. Benign melanocytic lesions included in the study were common acquired melanocytic nevi, intradermal nevi, juntional nevi and blue nevi.

Common melanocytic nevus

20 cases of melanocytic nevi were observed in the study, which constituted 70.7% of total melanocytic lesions. They have a varied presentation. 6 of them showed Plaque like presentation, 10 showed macules, 3 showed warty lesions and one was a pedunculated lesion. Histologically, these lesions showed proliferation of nevus cells in the form of nest and are classified according to location of nevus cells in relation to major epidermal landmarks. It is called as Junctional nevus if melanocytic proliferation is restricted to basal layer of epidermis. In intradermal nevus, all melanocytes were in dermis.

In 2 of the 20 cases (10%) of intradermal nevus observed in the study, nevus cells appeared spindle shaped, arranged in bundles, embedded in collagenous fibres forming neuroid tubes. This forms Neurotised nevus. **Neurotisation** is the tendency of melanocytes to adapt some of the phenotypic characteristics of Schwann cells, a spindle shaped elaboration of basement membrane material, and formation of structures called Pseudomeissnerian corpuscles.

Common Blue nevus

One case of common blue nevus observed in the study was located in forearm. Microscopically, Common Blue nevi were characterized by dermal proliferation of elongated, dendritic melanocytes. Junctional activity was absent. Common Blue Nevus resembles Nevus of Ota and Nevus of Ito, due to the presence of bipolar melanocytes. It was distinguished from them by the presence of sclerosis and dermal melanophages. It was distinguished from Dermatofibroma by the absence of hyperplastic epidermis.

Malignant melanoma

There were 6 case of Malignant melanoma observed in the study. All were nodular lesions, except for one, which was superficial spreading melanoma and was clinically thought as nevus prior to biopsy. Microscopically, all were characterized by prominent melanin pigmentation, junctional activity, invasion into surrounding tissue, marked cytologic atypia, nuclear grooves and inclusions, eosinophilic nucleoli and abundant mitosis. Level of invasion, depth of tumor thickness, tumor associated lymphocytes and number of mitosis were assessed for **prognastification** of tumours.

Clark system was used for assessing level of invasion.

i. Intraepidermal

ii. In the papillary dermis

iii. Filling the papillary dermis and stopping at the interphase between papillary and reticular dermis

iv. In the reticular dermis

v. In the subcutaneous fat

One of the six cases was in Clark's stage I , 3 were in stage III, one was in stage IV and one in stage V.

TABLE – 19

THESE OBSERVATIONS AS COMPARED WITH A PREVIOUS STUDY IN 2006 ON MELANOCYTIC SKIN LESIONS IN EGYPT (73) ARE GIVEN BELOW

	Hussein M.R. et al	**Present study**
Study period	1984-2004	2004-2006
Number of Benign nevus	12	21
Malignant Melanoma	21	6
Male : female ratio	1:2	1:1
Site	Extremities	Extremities
Age group		
Benign nevus	33+/- 5 years	25+/- 5 years
Malignant Melanoma	54+/- 5 years	42+/- 5 years
Clark level		
II	9%	16.7%
III	14%	50%
IV	38%	16.7%
V	38%	16.6%

Soft tissue Tumours

Tumours of fibrous tissue

Benign fibrous histiocytoma

Three cases of benign fibrous histiocytoma had been observed in the present study. Histologically, the lesion showed hyperplastic epidermis, elongation of rete ridges, separated by clear zone from the tumor in the dermis. The tumor showed fibroblast like spindle shaped cells, histiocytes and blood vessels in varying proportions. Pronounced epidermal hyperplasia, as noted by Schoenfeld. R.J. (64) was also observed in the present study.

Dermatofibrosarcoma Protruberans

Dermatofibrosarcoma Protruberans is a slowly growing dermal spindle cell neoplasm of intermediate malignancy. One case of Dermatofibrosarcoma Protruberans observed in a 33-year-old female in upper extremity in the present study, which is similar to observation by Guitierz. G. (21). Histologically the lesion was composed of densely packed, monomorphous, plump spindle cells arranged in a storiform pattern. The tumor infiltration into dermal stroma and subcutis was seen.

Due to the uniformity of spindle cells and storiform pattern, it was confused with benign fibrous histiocytoma, but attenuated epidermis with ulceration and infiltration into subcutis in the form of multilayered pattern pointed out the diagnosis of DFSP.

Soft Fibroma

Three cases of soft fibroma also called “fibroepithelial polyps” or “acrochordon” was observed in the present study. Two cases presented as multiple papules in neck region , one case was a solitary bag like pedunculated growth in trunk. Histologically it showed a flattened epidermis, overlying loosely arranged collagen fibers and mature fat in the center. They were usually excised for cosmetic reasons.

Keloid

Keloid usually follows injury, more common in presternal area. One case observed in the present study is a red raised firm lesion with smooth shiny surface. Histologically, the lesion showed collagen fibres in the granulation tissue, arranged in a whorl or nodular pattern. The nodules showed thick highly compacted, hyalinised bands of collagen lying in a concentric arrangement. The change observed by Murray. J.C. (50) was also seen in the present study.

Soft tissue vascular tumours

Vascular tumours in skin can be reactive, benign and occasionally malignant. The pathogenesis of these lesions remains a mystery. The presence of heterologous elements well developed smooth muscle and thrombosis supports hemangiomas.(29).

18 vascular tumours were observed in the study. Of which, 11 were capillary angiomas, 3 were cavernous hemangiomas, 3 were granuloma pyogenicum and one was glomus tumor.

Capillary Angioma

These tumours showed a lobular architecture and proliferation of endothelial cells. The endothelial cells were large, mitotically active and aggregated in solid strands. Presence of perineural invasion observed by Calonje (27) was not observed in the present study.

Granuloma Pyogenicum

Pyrogenic granuloma is a common proliferative lesion that often affects young adults of either gender, but the age range is wide. Hands, fingers and face are the common sites. These result of the present study were comparable to those of Patrick. S.J. (58).

Soft tissue- smooth muscle tumours

Cutaneous smooth muscle tumours present in three separate locations: arrector pilli muscles, blood vessel walls and genital or areolar skin. Benign and malignant smooth muscle tumours neoplasms may arise from each of these locations. Benign tumours exhibiting differentiation towards smooth muscle includes smooth muscle hamartoma and leiomyoma. Special stains like phosphotungstic acid hematoxylin, aniline blue, and Masson's trichrome are helpful in differentiating Muscle from collagen .

Leiomyoma

One case of solitary Leiomyoma was observed in the study. Arrectores pilorum gives rise to smooth muscle tumours in skin. This tumor was composed of interlacing bundles of smooth muscle fibres with which collagen fibres are intermingled. Multiple leiomyoma can be painful or sensitive to cold or touch. Granular and clear cell change as observed by Dobaski. Y (14) is not observed in the present study.

Neural tumours

Seven tumours with neural differentiation were observed in the present study. All were Neurofibroma. Four were single three were multiple. Histologically these lesions are circumscribed unencapsulated and composed of spindle cells with elongated wavy nuclei and are regularly spaced among thin, wavy collagenous strands.

Miscellaneous lesions

Cutaneous metastasis

Cutaneous metastases are of diagnostic importance because they may the first manifestation of an undiscovered internal malignancy. Two cases of cutaneous metastases were observed in the present study. In one case there was a nodule in perianal skin showing mucinous adenocarcinomatous deposit. The patient was found to have carcinoma colon. Other patient had anaplastic carcinomatous deposit in scalp that was lost to follow up.

Based on clinical diagnosis. 178 cases were benign and 22 cases were malignant. On histopathology 162 cases were benign and 38 were malignant.

Clinical Diagnosis was compared with Histopathological Diagnosis taking Histopathologic examination as the gold standard and sensitivity specificity test was performed.

TABLE - 20

COMPARISON AMONG TYPE OF NEOPLASM

Type of Neoplasm	Total	Clinically benign	Clinically Malig	HPE benign	HPE malig
Keratinocytic	106	88	18	79	27
Appendageal	29	29	0	27	2
Melanocytic	27	22	5	20	7
Soft tissue	38	38	0	36	2

Of the 106 keratinocytic tumors , 18 were clinically malignant, but on Histopathological examination 27 were found to be Malignant lesions. Appendageal tumors were all clinically Benign, but on Histopathologic examination 2 were malignant. Out of 27 melanocytic tumors, 5 were clinically malignant, on HPE 7 were malignant.

The histopathological examination of benign lesions increases the sensitivity of detecting malignant lesions at an early stage in all types of Neoplasm.

TABLE - 21

SENSITIVITY AND SPECIFICITY TEST

Clinical Diagnosis	**Gold Standard Test (HPE)**		**Total**
	Malignant	**Benign**	
Malignant	19	3	22
Benign	19	159	178

Sensitivity = 19/38 = 50%

Specificity = 159/162 = 98%

Positive Predictive Value = 9/22 = 86%

Negative Predictive Value = 159/178 = 89%

The sensivity of clinical diagnosis is only 50%, specificity is 98% and hence Histopathological examination of every skin tumour though they are clinically benign, becomes essential.

CONCLUSION

* Incidence of skin tumours in the present study is 6.13% out of all neoplastic lesions.

* Gender distribution - male: female ratio is 1.1:1.

* Most of the lesions were single (79%). Multiple lesions constituted 21%

* Of the various cutaneous tumours presented in this study, malignant lesions were only 19%, as compared to 81% of benign tumours.

* Most of the skin tumours were nodular lesions, followed by papular and macular lesions. Malignant tumours were seen more commonly in ulcerative lesions.

* Skin tumours were often found in head and neck region followed by extremities and rarely in trunk.

* **Excessive sunlight exposure was observed in 19.75% of patients with benign tumours and 60.5% of patients with malignant tumours.**

* **Clinical diagnosis helps in identifying patients into disease groups and histologic diagnosis, aids in management of patients.**

* **Clinical versus histopathologic correlation was positive in 86% of lesions.**

* Keratinocytic tumours were common in our study accounted for 53% of all tumours, of which 10.6% were benign tumours, 1.8% were pre malignant lesions, 31.13% were malignant tumours and 56.6% were tumor like lesions.

* **The incidence of Squamous cell carcinoma is increased when compared to other cutaneous malignant tumours.**

* There were no variants of Squamous cell carcinoma noted in this study.

* The diagnoses of malignant skin tumours were straightforward and problems were encountered in adnexal tumours.

* Although histopatholgy remains the gold standard for most dermatologic diagnosis, it must be recognized that not all lesions are amenable to definitive specific diagnosis.

* To make appropriate histologic diagnosis and to provide an estimate of prognosis in most skin lesions, ultrastructural immunohistochemical and molecular aids can increase the specificity.

* Another advanced measure of the biological relevance of a diagnostic system is through the correlation of traditional diagnostic labels with molecular study. These correlations can be expected to increase in the future while strengthening the relationship between our present empirically based classification schemes and the underlying biology.

- **It is likely that the best approximation to the goal of improving diagnostic specificity will be achieved by a detailed correlation of findings at molecular, histologic and gross anatomic levels with the physical findings and clinical history interpreted in the context of the whole patient and his or her environment with long term follow up serving as the gold standard.**

BIBLIOGRAPHY

I. Annie Varghese, An Analysis of 128 cases of skin cancer, M.D. thesis ; Madras University 1967.

II. Berman A, Winkellmann R K , Inflammatory Seborrhic Keratosis with mononuclear cell infiltration. J Cutaneous Pathol 1978; 5: 353.

III. Brand D, Ackermann 2000, Squamous Cell Carcinoma not Basal Cell Carcinoma is the most common Cancer in Humans ; J Am Acad Dermatol 42: 523-526.

IV. Brodin B, Mehregan A H, VErrucous Carcinoma; Arch Dermatol 1980; 116:987.

V. Brownstein M H . Shapiero L , Pilosebaceous Tumors ; Int J DErmatol 1977:16 340-352.

VI. Burg et al WHO/ EORTC Classification of Cutaneous Lymphomas 2005. Histological and Molecular aspects. J Cutaneous Pathol 32

VII. Byers HR et al ' Pathologic Parameters in the Diagnosis and Prognosis of Primary Cutaneous Melanoma' , ; Hematol Oncol Clin North America 1998 Aug, 12(4):717-35.

VIII. Cannon J R, Schneider ; Recent Developments in Adnexal Pathology in Moschella SM Dermatol Update, NewYork , Elseiver Publisher 1982:236.

IX. Carrow A, Brownstein BR, Tumors of Skin , in Moschella SL, Hurley HJ eds; Dermatology, 2nd edn , WB Saunders Company, Philadelphia 1996; 1721.

X. Chakravarthy RC. Malignant Neoplasms orf skin in Eastern India; Indian J Cancer 1968; 5; 133-144.

XI. Clark WH, elder D.E, 'A study of Tumor Progression: the Precursor lesion of Superficial spreading and Nodular Melanoma' , Human Pathol 1984: Dec 15(12) 1147-565.Conway H, Hugo NE, Tulenkov F. Carcinoma of skio, in surgery of Tumors of skin 2 nd edn . Charles C. Thomas. 1966; 165

XII. Criber B et al; Tumors arising in Nevus Sebaceous, A study of 586 cases; J Am Acad

Dermatol 2000; 42(2) 263-268.

XIII. Dignostic Index and Tumor Registry of New York Hospital. Cornell Medical Centre; 1963; 3.

XIV. Dobashi Y, Iwabuchik , Nakahatha J et al; Combined Clear and Granular cell Leiomyoma

XV. Doca AF, Cutler S. J. , morbidity from Cancer in United States, Public Health Monograph, Public Health Services publication 1959: 56:590.

XVI. Eller JJ. General Considerations in Tumors of DSkin 1 st edn Lea and Feliger, Philadelphia 1939; 13.

XVII. Ewing J, Modern attitude towards traumatic Cancer Arch Path 1985: 19: 690-728.

XVIII. Feldmann A H et al Clear Cell Hidradenoma ; A review of Literature ; J Foot Ankle Surg 1997; 36: 21-25.

XIX. Fisher B. K., mac pherson M, Epidermal Cyst of Sole. J Am Acad Dermatol, 1986; 15: 1127.

XX. Gray hR, Helwig EB, E pithelioma Adenoides Cysticum and Solitary Trichoepitheloima; Arch Dermatol 1963:87:102.

XXI. Guteirez G et al , Dermatofibroma Protruberens : Int J Dermatol 1984; 23:396.

XXII. Hashimoto K, Fisher B K in Lever Histopathology of Skin 9 th edn Steatocystoma.

XXIII. Headington J T. Mixed Tumors of Skin: Eccrine and Apocrine Types arch Dermatol 1961; 84: 989.

XXIV. Helwig E B , Classifuication of Skin tumors, in A Collection of Papers presented at 20 th annual Clinical Confernce on Cancer 1995, Neoplasms of Skin and Malignant Melanoma, Year Book Medical Publishers ; 57.

XXV. Hervella et al 'Vascular Tumors as a syndromic Indicators' St Sanit Navas 2004 : 27 suppl 33-44.

XXVI. Hirsch P Helwig E B. Chondroid Syringoma; arch Dermatol 1961; 84: 835.

XXVII. Hunt SI et al , Vascular tumors of Skin, a selective review Semin

XXVIII. iagnostic Pathol 204 Aug 21(3): 166-218.

XXIX. Hunt SJ et al Microvascular Hemangioma J cutaneous Pathol 1991: 18; 235.

XXX. Hussein etal , ClinicoPathological Features of Melanocytic Skin lesions in Egypt; Eu J cancer Prev 2006 Feb; 15(!); 64-68.

XXXI. Hussein MR ' Melanocytic Dysplastic Nevi occupy the middle ground between Benign melanocytic Nevi and Cutaneous Mlignant Melanomas emerging clues,' J Clinical Pathol 2005 May 58(5):453-6.

XXXII. Jayalakshmi et al Cutaneous Adnexal Neoplasms in biopsy Specimen Processed in Dept of pathology, University of Malayaa ; Ann Acad Med Singapore196; 25(4): 522-525.

XXXIII. Jinbow K et al 'The epidermal Melanin Unit in the Pathophysiology of Primary Cutaneous Melanoma' Am J Dermatopathol 1991; Apr; 13(2) 179-88.

XXXIV. Johnson DE, Basal Cell Epithelioma of Palm, Arch Dermatol 1960; 82 ;253.

XXXV. Johnson W C, ' Pathology of Cutaneous Vascular Tumors ' Int J Dermatol 1976 May 15(4) 239-70.

XXXVI. Joost J Van den Oord et al ; Perivascular Spaces in eccrine Spiradenoma ; A clue to histological Diagnosis; AmJ Dermatopath 1995, 17(3) 266-270.

XXXVII. Julien LG, Bover PW , A clinical review of 209 pilomatricomas; J Am Acad Dermatol1998: 39, 191-195.

XXXVIII. Ken Hashimotos, Tumors of Skin Appendages in Fitzpatrick Dermatology in General Medicine, Fitzpatrick TB 5 th edn Mc Grawhill 1999; 890-914.

XXXIX. Kerl h. What is the boundary that separates a thick Solar Keratoses and a thin Squamous cell carcinoma. Am J DErmatopathol 1984: 6; 305.

XL. Komu J. Wateanbe S. Desmosome like structures in the cytoplasm of normal Keratinocytes. Arch Dermatol 1975: 245-402.

XLI. Levi F, Br J Cancer 1995 oct; 72(4) 1047-53, Trends of Skin Cancer in Carton of Vaud, 1976-92.

XLII. Mac Donald EJ. Bubendolf E; Some epidemiological aspects of Skin Canncer in Tumors of Skin ; Year Book Medical Publishers Chicago 1964; 23-65.

XLIII. Mac Donald EJ. Etiology of Skin Cancer. J invest Dermatology 1959; 32: 379-385.

XLIV. Mackie RM , Tumors of skin, in Rook, Wilkinson DS, Ebling FJG, Burton J eds: textbook of Dermatology, 6th edn, Oxford University Press1998, 1651.

XLV. Maize J.C. 'Dysplastic Melanocytic nevi in histologic association with primary Cutaneous melanoma'

XLVI. Manchini et al ' Histogenesis of Experimentally Produced Ke;loids 1962, Journal inv Dermatol 38: 143-181.

XLVII. Marie L J, Stern RS , Prevalence and Ecology of skin Disorders in Fitzpatrick TB, Eisen AZ, WOolff K, Anstein KF ' Dermatology in General Medicine' 2nd edn Mc Grawhill Book Company , New york 1979 ; 3 .

XLVIII. Mathews et al ' Cutaneous Leiomyoma with cytologic atypia' Dermatol Surg 2004 Sep:30(9) 1249-51

XLIX. Meittares et al ' Evaluation of Biologic P{otential of Smooth Muscle Tumors' Histopathology 2006 Jan 48(1) 97-105. PMID 1635954.

L. Murray JC et al Keloids: A rerview J Am Acad Dermatol 1981: 4; 461

LI. Oslon R. L. Nordquist K. Dyskeratosis in Bowens disease int Dermatol 1969; 81: 676.

LII. Pack Gt, Le Fevel RG, age, sex distribution and incidence of neoplastic Diseases at memorial Hospital N.Y.C. with comments on cancer Ages. J. Can Res 1930; 14 167-190.

LIII. Patrick SJ , Wissk Mulliken JB, Pyogenic Granuloma (Lobular Capillary Hemangioma)

A ClinicoPathologic study of 178 cases. Paediatric Dermatol 1994: 8: 267.

LIV. PayMaster JC., Talwalkar G V, Gnagadharan P. Carcinoma and Malignant Melanoma of Skin in Western India ; Royal Coll Surg 1971 :16: 166-173.

LV. Philip E Heboit et al WHO Classification of Skin Tumors , Pathology and Genetics of Skin Tumors, IARC Press LYON 2006.

LVI. Qui JH et al Zhonghua Zhong Lin ZA Zhi, 1986 Jan 8(1) 48-50, Pathological Studies on epidermic and appendageal Tumors and Tumor like Lesions- analysis nof 3425 cases.

LVII. Rager EL, Bridgeford E P et al ' Cutaneous Melanomas: update on Prevention, Screnning, Diagnosis and Treatment' ; Am Fam Physician 2005, Jul 15 72(2) 269-76.

LVIII. Raj S, Calonje E. 'Cutaneous Pilar Leiomyoma- Clinicopathologic Analysis of 53 lesions in 45 Patients . Am J Dermatopathol 19:2-9.

LIX. Redd D j, Venkateshwara Rao, Malignant neoplasms of Skin , Indian J Dermatol Veneral Lepr 1964; 30; 44-54.

LX. Requena et al ' cutaneous Vascular Proloferations Part II hyperplastic and Benign Neoplasms. J Am Acad Dermatol 1997 Dec 37(6) 887-919.

LXI. Rhodes A.R. Melanocytic Precursors of Cutaneous Melanoma, estimated risks and guidelines for management. Med Clinics North America 1986 Jan 70 (1) 3-37.

LXII. Richard B. Odon, Williams D James, Timoti G Berger, Andrew Diseases of Skin 9th edn W. B Saunders; newYork 2000; 850-867.

LXIII. Rippey J J , Whiting Da, Moles and Melanomas; Lancet 1977 , Jul (16) 137 PMID 69215.

LXIV. Schonfeild RJ, Epidermal Proloferatons Ovrlying Histiocytomas; arch Dermatol 1964: 90; 266.

LXV. Scolyer RA et al Pathology of melanocytic Lesions: New Controversial and Clinically important issues; J. Surg Oncol 2004 jul: 86(4) 200-11.

LXVI. Shaffer et al ‘ Keloidal Scars a Review with a critical look at Therapeutic Options’ J Am Acad Dermatol 2002 46; s63-s97.

LXVII. Smith J D, Chernowsky M E.; Hidrocystoma, Arch dermatol 1973; 108:676.

LXVIII. Solanki A L. Anand V. K., gupta k, Neoplasms of Sweat Glands; Indian J Derematol venerol 1989; 55: 108-113.

LXIX. Solanki A L., Anand U K, Gupta K. Neoplasms of Sweat Glands; Indian Journal of Dermatol Vernerol 1989; 55, 108-113.

LXX. Solanki AL Anand VK. Gaur SK, Arora AL. Neoplasms of Hair Follicle, Indian J Dermatol Venerol lep 1989; 55:33-37.

LXXI. Sommer S C. Mac Mannus RG, Multiple arsenical Cancers of Skin and Internal Organs . Cancer 1953 ; 6: 347-359

LXXII. Trevas W pack GT, The Development of Cancetr in Burn Scar. Surg Obst Gynaec 1930; 57-749-82.

LXXIII. Vaishnav P Dharkar DD. Adnexal Tumors of Skin ‘ Indian J Pathol Bacteriol 1974; 1733-38.

LXXIV. Ward G E, Hendrick J W, Diagnonosis and Treatment of Tumors of Head And Neck , Baltimore 1950 as Quoted by Hebert et al.

LXXV. Watson T A , Incidence of Multiple Cancer, Cancer 1953; 6 365-371.

LXXVI. Weigh et al , Appendageal Tumors IADVL : National Conference Jan 2002:258.

LXXVII. Weinstock M A et al 1997, Death from Skin Cancer among elderly: Epidemiological Patterns; Arch Derrmatol 133: 1207-1209.

LXXVIII. Williamze R et al 1997 , EORTC Classification of primary cutaneoyus Lymphomas, A proposal from the Cutaneous Lymphoma Study Group of European Organisation for Research and Treatment of Cancer. Blood 90: 354-371.

LXXIX. Yahoob et al ,’ Specdtrum of Cutaneous Appendage tumors at Agha Khan University Hospital’ ; J Pak Med Assoc 2003 sep; 53(9) 427-31.

APPENDIX –I

Staining Methods

Hematoxylin and Eosin

Harris Hematoxylin Preparation

Hematoxylin	-	2.5 g
Absolute Alcohol	-	25 ml
Pot Alum	-	50 g
Distilled Water	-	500 ml
Mercuric oxide	-	1.25 g
Glacial Acetic Acid	-	20 ml

Dissolve the Hematoxylin in Alcohol; separately dissolve Pot Alum in warm distilled water. Mix the two and heat. Add the mercuric Oxide, when it comes to boiling. Rapidly cool the stain in water. Add Glacial Acetic Acid to cooled stain.

Eosin Preparation
Eosin X 10 g
Distilled water 50 ml
95% Alchohol 950 ml

- Deparaffinise the sections,
- Bring the sections to water
- Hematoxylin for 5 min
- 1% Acid Alcohol 2 dips
- Wash in Ammonia Water or Saturated Lithium Carbonate
- Eosin 3 dips
- Clear in Xylol
- Mount in DPX.

Results : Nucleus – Blue
Cytoplasm- pink

Periodic Acid Schiff Stain (PAS)

Solution

a) 0.5% Periodic Acid Solution
b) Schiff Reagent
   Basic Fuschin-1gm
   Distilled Water- 200 ml
   Potassium Metabisulfite 2 g
   1 N HCl 10 ml
   Activated Carbon 0.5 g

   Dissolve the Basic fuschin in hot distilled water. Bring to boiling . Then cool it and add Pot metabisulfite and Hcl. Let it bleach for 24 hrs.
   Then add the Activated Carbon and Shake it for a minute
   Repeatedly filter till colourless.

Staining Method

- Bring the sections to water
- Place the slide in 1% Periodic Acid Solution for 5 min
- Wash in Distilled Schiff's Reagent for 15 min
- Wash in Distilled water
- Harris Hematoxylin for 25 min
- Wash in Tap water
- Differntiate in 1% Acid Alcohol
- Wash in Running tap water(Blueing)
- Clear and Mount in DPX.

Results : Glycogen: Magenta
Nucleus : Blue

Vangieson Stain

1. Bring the sections to water
2. Treat with Picrofuchsin Stain (1cc 1% Acid Fuchsin and 9 cc of Sat Picric Acid) and leave it for 15 min
3. Dip in Distilled water.
4. Blot and Dry and Mount

Result: Muscle- yellow
Fibers- Orange

APPENDIX – II

PROFORMA

Serial No.

Biopsy No.

Date

Clinical Parameters

Name

Age

Sex

IP No.

Occupational history

H/O Excessive Sunlight Exposure

Site of the lesion

Clinical nature of lesion

Clinical Diagnosis

Histopathologic Examination

Histopathologic Diagnosis